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CENTER FOR DISEASE CONTROL

INFLUENZA - RESPIRATORY DISEASE SURVEILLANCE

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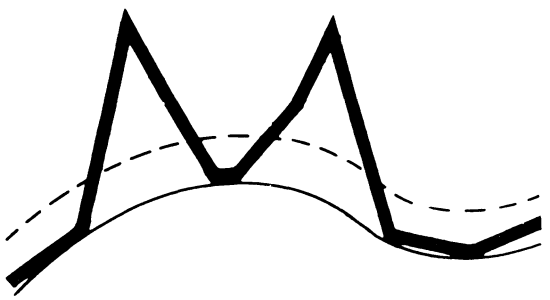

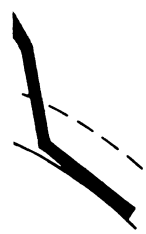

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PUBLIC HEALTH SERVICE

PREFACE

Summarized in this report is information received from State Health Departments and other pertinent sources, domestic and foreign. Some of the information is preliminary. It is intended primarily for the use of those with responsibility for disease control activities. Anyone desiring to quote this report should contact the original investigator for confirmation and interpretation.

Contributions to the surveillance

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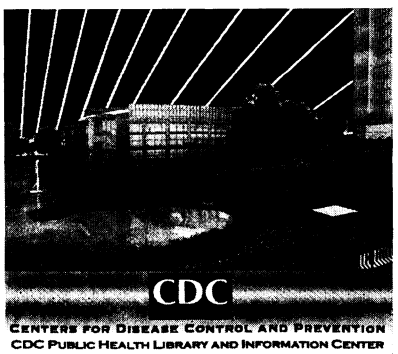

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Center for Disease Control . . . . . David J. Sencer, M.D., Director

Bureau of Epidemiology . . . . . Philip S. Brachman, M.D., Director

Viral Diseases Division . . . . . Michael B. Gregg, M.D., Chief

Respiratory Diseases Surveillance Unit . . . . . Michael A. W. Hattwick, M.D., Chief
Lawrence Corey, M.D.
Robert J. Rubin, M.D.

Statistics Activity . . . . . W. Jere Housworth, Chief

In Collaboration With:

Bureau of Laboratories
Respiratory Virology Section . . . . . Marion T. Coleman, Ph.D., Acting Chief

World Health Organization
International Influenza Center
for the Americas, CDC . . . . . Walter R. Dowdle, Ph.D., Director

INFLUENZA SURVEILLANCE 1972-1973

I. SUMMARY

From September 1972 to September 1973, influenza reached epidemic proportions in major areas of the United States. As in the 1971-1972 influenza season, widespread outbreaks occurred in a number of states.* In nearly all influenza A outbreaks, a virus similar to A2/England/42/72 (H3N2) was demonstrated to be the responsible agent. Regionally, the greatest morbidity and mortality was seen in the Pacific, Mountain, Middle Atlantic, East North Central, and West South Central regions. Nine states reported localized outbreaks of influenza B: Arizona, California, Colorado, Hawaii, Idaho, Minnesota, Oregon, Washington, and Wisconsin. In 3 of these (Hawaii, Washington, and Minnesota) hemagglutinin inhibition tests revealed an antigenic shift from previous B isolates in the United States. This report summarizes the morbidity and mortality data received by the Center for Disease Control (CDC) for the 1972-1973 influenza season.

II. SURVEILLANCE METHODS

A. Case Reporting

Influenza is not a nationally reportable disease by cases although states are requested to report outbreaks to CDC. Nevertheless, approximately 25 states currently report cases of influenza or influenza-like disease to CDC and publish them in their own morbidity reports.

B. Telephone Reporting System

In previous years, CDC conducted periodic telephone surveys of state epidemiologists during the fall and winter season, to assess the progress of influenza activity. The data obtained varied considerably from state to state but was useful in providing a rapid general assessment of influenza activity throughout the United States.

In 1972 and 1973, in an endeavor to develop more uniform data on a nationwide basis, CDC enlisted the cooperation of state and territorial epidemiologists and laboratory directors to provide information routinely about: 1) emergency room visits to large community hospitals in major cities within their states, 2) school and industrial absenteeism, 3) number of specimens submitted for viral isolations, and 4) the number of viral isolations made by each laboratory.

In 1972-1973 this information was telephoned to the regional offices of CDC weekly by 44 states (Table 1), and over 60 cooperating laboratories forwarded data weekly to the Influenza Center for the Americas in Atlanta.

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*CDC classifies the extent of influenza in 4 categories: 1) isolated cases, 2) isolated outbreaks, 3) regional involvement (outbreaks recognized in contiguous counties, but altogether involving counties comprising less than one-half of the state's population, and 4) widespread involvement (more than half of the counties or more than half of the population).

DHEW Publication No. (CDC) 74-8207

Table 1

Participation in the National Influenza Surveillance System

Region	State	City/County
I	Connecticut	Hartford
	Maine	Augusta
	New Hampshire	Concord
		Portsmouth
	Rhode Island	Providence
II	New York	Buffalo
		Albany
		Syracuse
		New York
III	Delaware	Kent County
		New Castle County
		Sussex County
	Maryland	Baltimore
	Pennsylvania	Philadelphia
		Pottstown
		Hazelton
		Allentown
		Wilkes Barre
		Scranton
		Lewiston
		Williamsport
		Pittsburgh
	Virginia	Norfolk
		Portsmouth
		Charlottesville
		Richmond
		Alexandria
		Roanoke
	Washington, D.C.	
	West Virginia	Statewide
IV	Alabama	Montgomery
	Florida	Hillsborough County
	Georgia	Atlanta
	Kentucky	Frankfort
	Mississippi	Jackson
	North Carolina	Chapel Hill
		Orange County
	South Carolina	Anderson County
		Richland County
	Tennessee	Nashville
		Memphis
V	Illinois	Springfield
	Indiana	Indianapolis
	Michigan	Lansing
		Detroit
	Minnesota	St. Paul
	Ohio	Columbus
	Wisconsin	Madison
VI	Arkansas	Little Rock
	Louisiana	New Orleans
		Monroe
	New Mexico	Albuquerque
	Oklahoma	Oklahoma City
	Texas	Amarillo
		Austin
		Beaumont
		Brownsville
		Corpus Christi
		Dallas
		El Paso
		Fort Worth
		Houston
		Lubbock
		San Antonio
		Tyler
VII	Nebraska	Lincoln
		Omaha
VIII	Colorado	Denver
	Montana	Helena
	North Dakota	Grand Forks
		Fargo
	South Dakota	Pierre
	Utah	Salt Lake City
	Wyoming	Cheyenne
IX	Arizona	Phoenix
		Yuma
	California	San Francisco
		Oakland
		Los Angeles
	Hawaii	Honolulu
	Nevada	Washoe County
		Las Vegas
X	Alaska	Anchorage
	Oregon	Portland
	Washington	Seattle
		Spokane

C. Mortality Reporting

Deaths are reported to CDC each week by the Vital Statistics Offices of 122 United States cities and are published in Table IV of the Morbidity and Mortality Weekly Report (MMWR). They are listed by place of occurrence of death, and include deaths of persons whose residence may have been elsewhere, but do not include deaths of residents which occurred in other vital statistics jurisdictions. The report is a count of death certificates filed each week, and may include some deaths which occurred in preceding weeks. The number of delayed certificates usually increases during holiday periods, causing a drop in the number of deaths reported for the holiday week, followed by an increase when the delayed certificates are included in the report for the succeeding weeks.

This information reflects influenza activity by a rise in mortality usually 2-4 weeks after the clinical disease is noted to be widespread. These data provide the best available epidemiologic evidence of the extent and severity of an epidemic in the country as a whole.

Expected mortality is determined by the use of data for prior years to predict the weekly mortality level for the coming year (1, 2, 3). The method works well in general because the same seasonal pattern is observed each year, the same peaks are observed in years with little influenza activity, and the same nadirs are observed almost every year. The exception to this observation is that over a period of years there is sometimes a general upward or downward trend in mortality. Except for infant mortality, this trend has not been very great in recent years.

The expected mortality level is determined by using weekly data for the previous 4- or 5-year period, omitting data for epidemic periods, and fitting the data to the following model by least squares:

$$\hat{y} = u + rt + A_1 \cos\frac{2\pi t}{52} + B_1 \sin\frac{2\pi t}{52} + A_2 \cos\frac{4\pi t}{52} + B_2 \sin\frac{4\pi t}{52}$$

The expected level is obtained by inserting the appropriate value of t in the equation where t is the number of the week from the beginning of the data which was fitted to the model. This procedure allows for a general mean, a slope, and annual and semi-annual cycles in the data, and omission of epidemic data prevents an inflation of the expected level during the influenza season.

Charts are prepared in which the reported numbers of deaths are shown as dots joined by line segments. The solid line for each mortality category is the expected number of deaths. The dashed line is the "epidemic threshold," a criterion for the recognition of significant deviations in excess of the expected number (1, 2, 3).

The charts are drawn to a scale that allows the distance between the expected and threshold levels to be constant for every curve. This device allows one to compare the influenza activity between regions by glancing at the regional chart. Although the vertical labels are different, comparison of the absolute distance on the chart between observed and threshold levels between regions shows whether the mortality is significantly higher in one region than another.

References

1. Collins SD, Lehmann J: Excess deaths from influenza and pneumonia and from important chronic diseases during epidemic periods, 1918-1951, Public Health Monogr 10 (PHS Publication 213), US Government Printing Office, Washington, DC, 1953

2. Serfling RE: Methods for current statistical analysis of excess pneumonia-influenza deaths: Public Health Rep 78:494-506, 1963

3. Serfling RE: The current mortality chart. Morbidity and Mortality Weekly Rep 14(1):8-11, 1965

III. RESULTS OF 1972-1973 SURVEILLANCE

Mortality in 122 cities due to all causes for the 1972-1973 influenza season is shown in Figure 1. Pneumonia and influenza deaths were significantly elevated above the epidemic threshold from the 1st to the 10th week of 1973 (Figure 2). Although influenza was reported throughout the country, excess mortality was mainly limited to the Pacific, East North Central, Middle Atlantic, and West South Central regions.

Fig. 1 MORTALITY IN 122 UNITED STATES CITIES

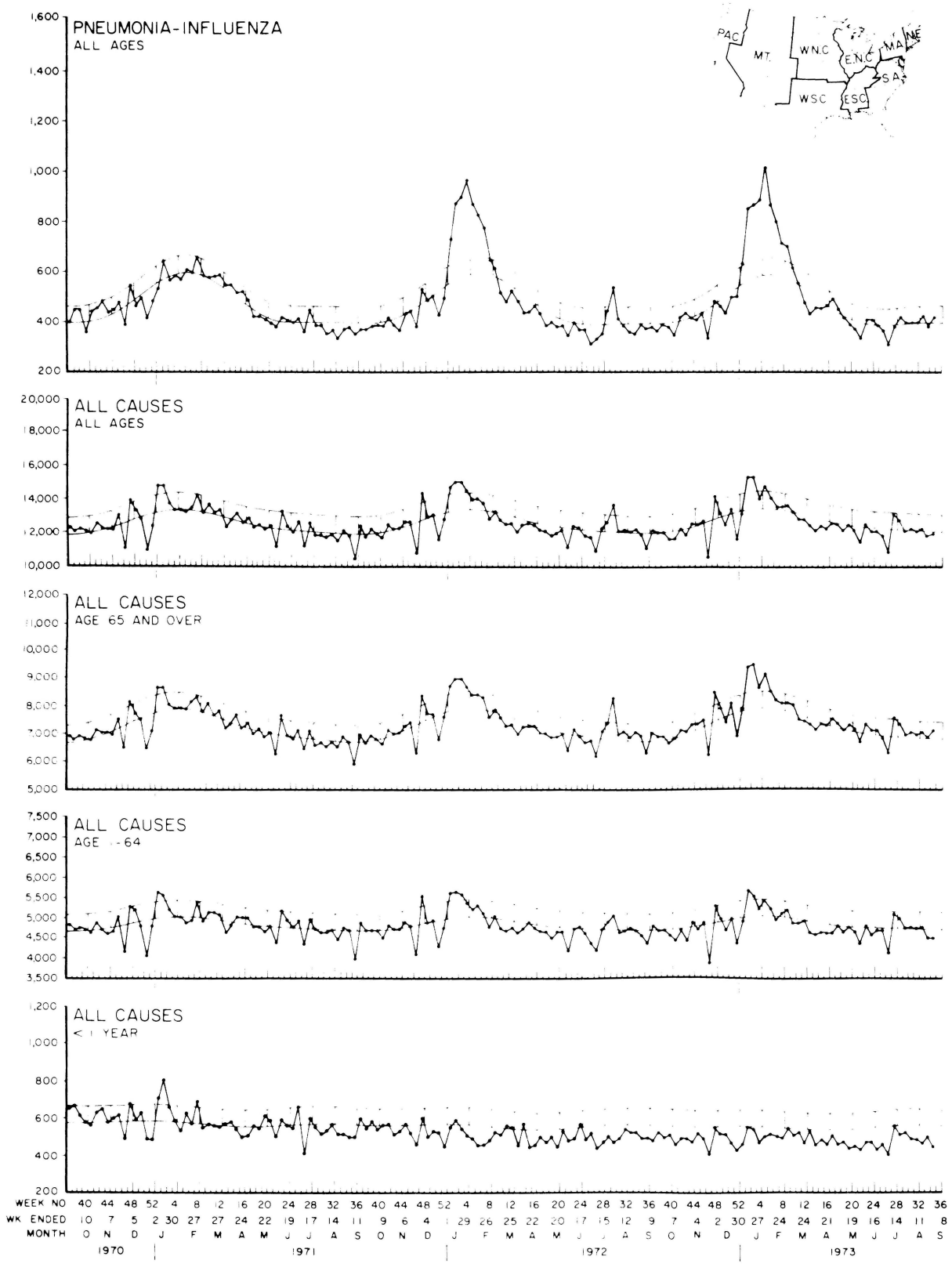
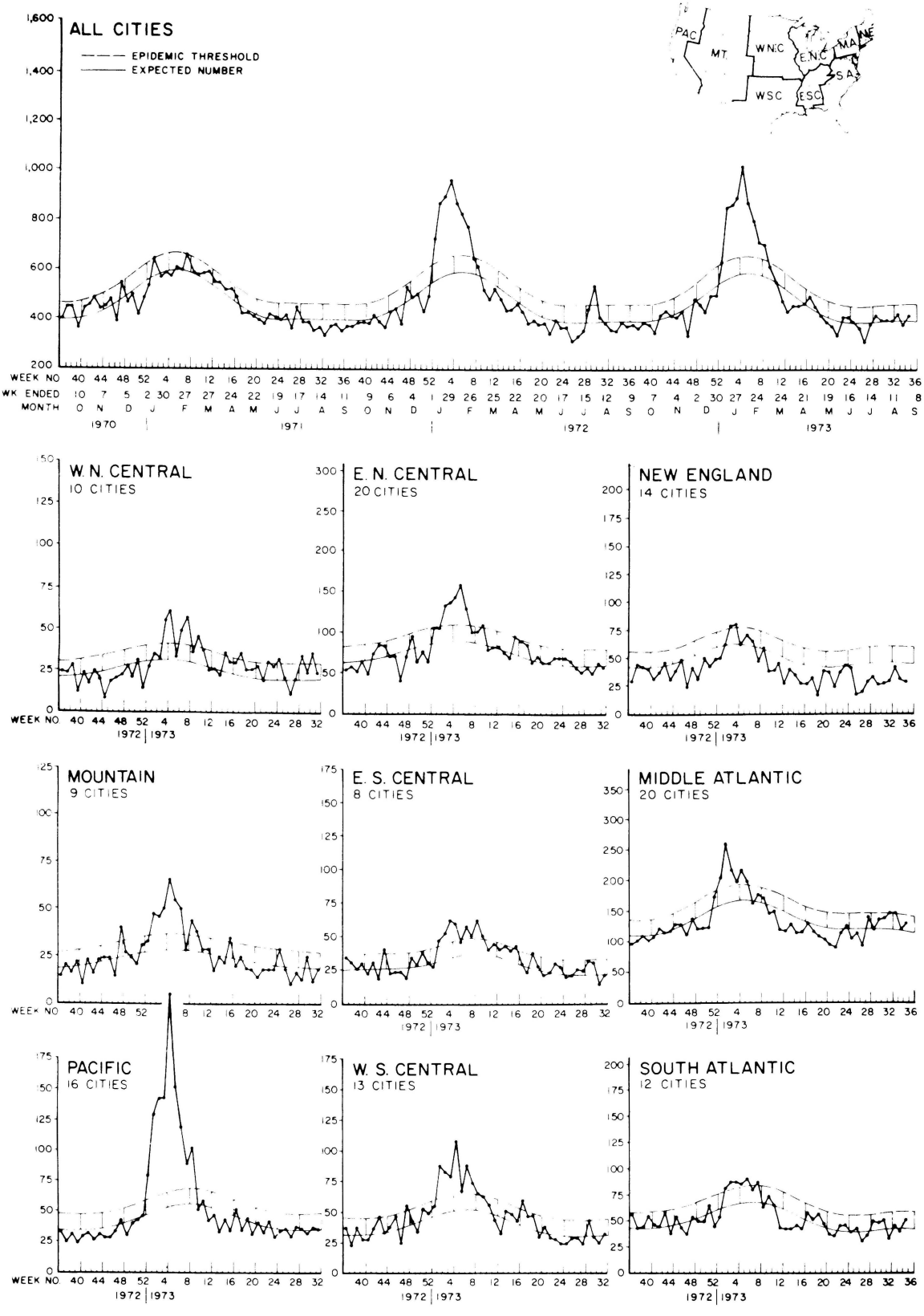


Fig.2 PNEUMONIA-INFLUENZA DEATHS IN 122 UNITED STATES CITIES



Excess mortality from pneumonia and influenza for 1972-1973 in the 122 United States cities was approximately 2,200. This total, drawn from an urban population of approximately 70 million persons, represented the largest number of excess deaths since 1968-196

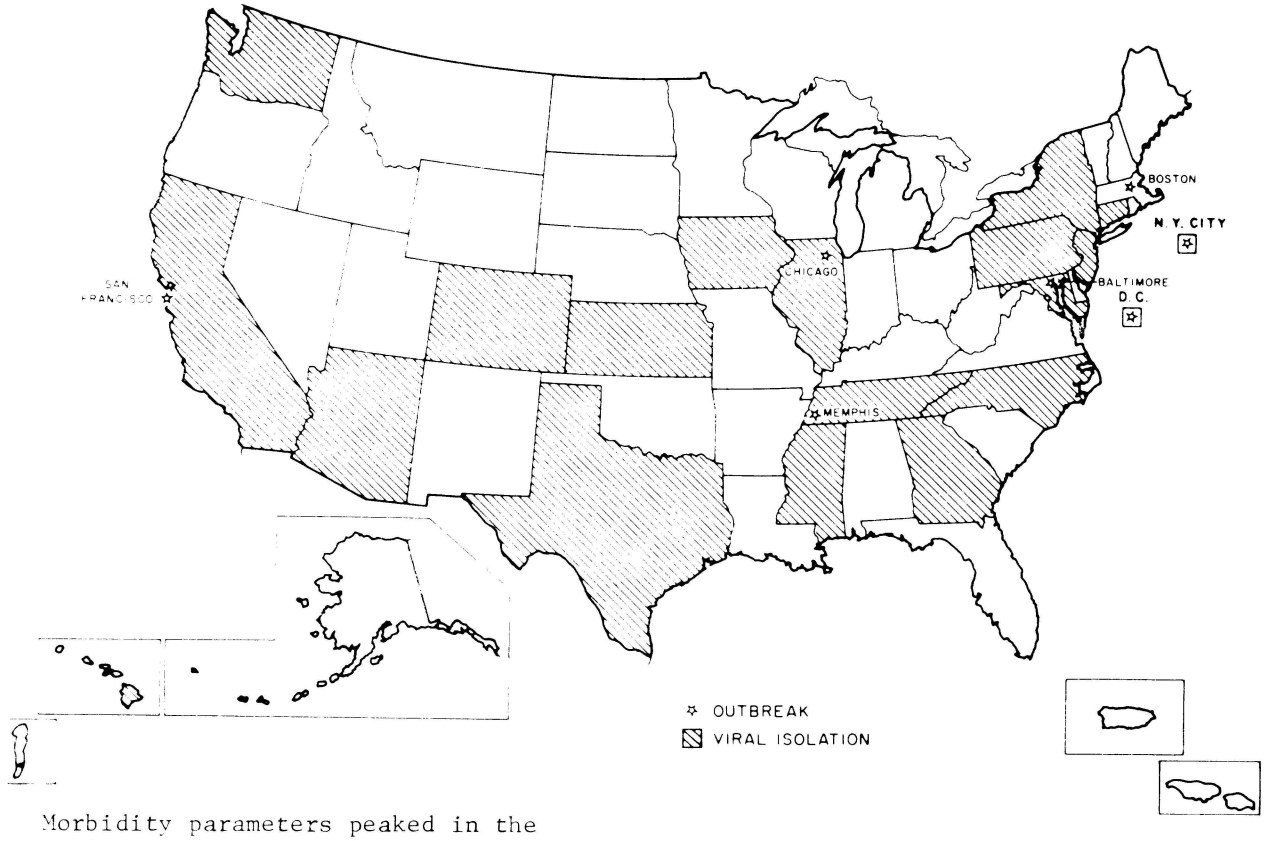
The first reported influenza activity in the United States in the 1972-1973 season occurred in 2 military installations in Colorado. The first outbreak occurred between October 21 and November 4, 1972, at the United States Air Force Academy in Colorado Springs. The clinical attack rate for the 2-week period was 211 per 1,000 cadets; a total of 870 cadets reported to sick call during the outbreak. Subsequently, during the week ending November 4, 1972, there was an outbreak of influenza at Lowry Air Force Base near Denver. In late November and early December, 2 additional military installations reported outbreaks -- Fort Huachuca in southeastern Arizona, and Ent Air Force Base near Colorado Springs.

During the last week of November 1972, the first major civilian outbreak of influenza was noted in Baltimore, Maryland. This outbreak was heralded by a 20-30% increase in emergency room visits at 2 hospitals participating in a surveillance system conducted by the state of Maryland and CDC. Confirmation of the outbreak was obtained by isolations of the virus in both Maryland and CDC laboratories. During the last week of November and the first few weeks of December, CDC received reports of isolated outbreaks of influenza-like disease from Pueblo, Colorado, 2 colleges in Massachusetts, and a job corps training center in Pennsylvania. By December 23, 1972, influenzavirus had been isolated from 14 states, with significant outbreaks in New York City, Baltimore, and the San Francisco Bay area (Figure 3).

Fig 3 INFLUENZA SURVEILLANCE, UNITED STATES, DECEMBER 23, 1972

By January 13, 1973, influenzavirus had been isolated in 18 states: Arizona, Connecticut, Massachusetts, New Jersey, New York, Georgia, North Carolina, Colorado, Kansas, Texas, Pennsylvania, Maryland, Washington, Illinois, California, Tennessee, Hawaii, and Iowa. Outbreaks of influenza were reported in New York City, Boston, Chicago, Memphis, the Baltimore-Washington, D.C. area, and in the San Francisco Bay area (Figure 4). The rest of the country was reporting only sporadic cases.

Fig. 4 INFLUENZA SURVEILLANCE, UNITED STATES, JANUARY 13, 1973



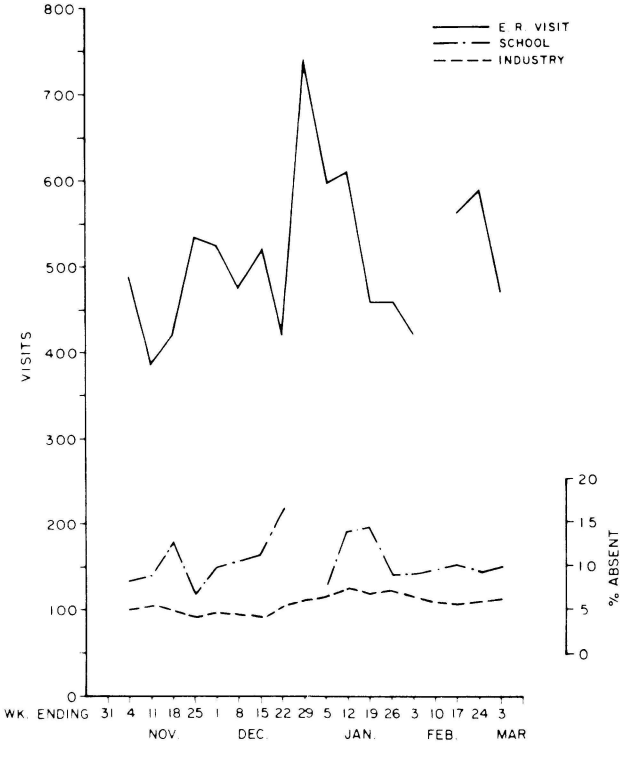
Morbidity parameters peaked in the mid-Atlantic and Pacific areas in early January 1973. As noted earlier, excess mortality exceeded the epidemic threshold for 2 or more consecutive weeks in both these areas in mid-January. By the end of January, morbidity from influenza was decreasing in the northeast but states in the southeast and mid-west were reporting increasing numbers of cases of influenza. Influenza-like disease peaked in these areas in mid-February. By the first week of March 1973, morbidity indicators had returned to normal in all reporting areas.

Representative State Results

Representative examples of the emergency room and school and industrial absenteeism monitoring system used by state epidemiologists are summarized below:

California: Influenza activity in northern California began to increase the 3rd week in December and continued through January. This increased activity is reflected in the number of emergency room visits reported by a hospital in the San Francisco area (Figure 5). However, neither school nor industrial absenteeism

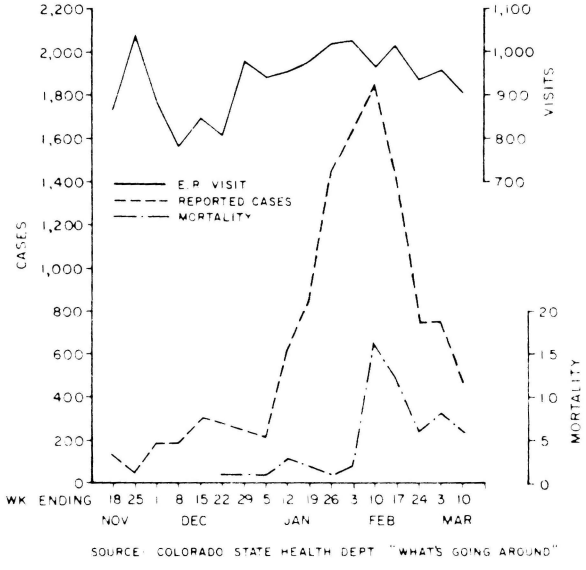
Fig. 5 EMERGENCY ROOM VISITS AND SCHOOL AND INDUSTRIAL ABSENTEEISM, CALIFORNIA (SAN FRANCISCO AREA), NOV. 4, 1972 — MAR. 3, 1973



increased above their normal seasonal levels in the San Francisco Bay area, thus demonstrating the occasional insensitivity of school and industrial absenteeism as indices of influenza activity.

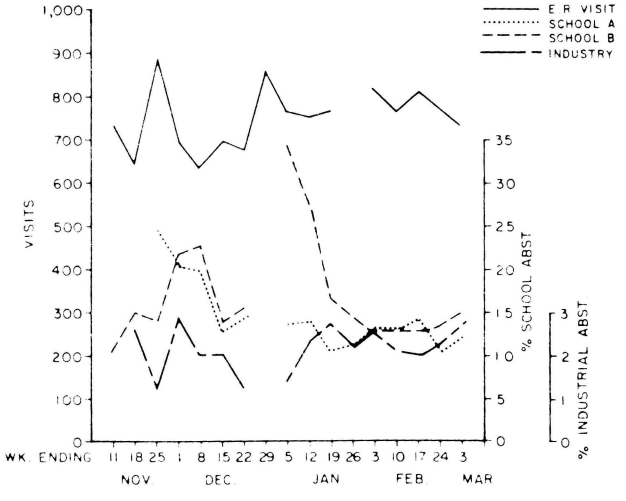
Colorado: The results obtained by Colorado from influenza surveillance within the state point out an important problem in influenza surveillance. Influenza and pneumoni mortality, cases of influenza reported by private physicians, and emergency room visits in Denver hospitals were monitored (Figure 6). While both the number of reported cases of influenza and influenza mortality increased during late January and early February, emergency room visits remained the same. This can be explained by the fact that while most of Colorado was affected by influenza in late January and early February, the Denve area, where emergency room surveillance was centered, did not experience influenza until later in the year. Figure 7, which represents surveillance within the city of Denver, shows essentially no increase in either emergency room visits or school and industrial absenteeism during the period under surveillance.

Fig. 6 EMERGENCY ROOM VISITS, REPORTED INFLUENZA CASES, AND INFLUENZA MORTALITY, COLORADO, NOV. 1972–MAR. 1973



SOURCE: COLORADO STATE HEALTH DEPT. "WHAT'S GOING AROUND"

Fig. 7 EMERGENCY ROOM VISITS AND SCHOOL AND INDUSTRIAL ABSENTEEISM, DENVER, COLO., NOV. 21, 1972 - MAR. 3, 1973



Ohio and Illinois: The correlation between emergency room visits and industrial and school absenteeism was illustrated in both Ohio and Illinois. In Columbus, Ohio, emergency room visits peaked during the last week in January, while industrial absenteeism rose from 3% to 5.5% during the month (Figure 8). In Springfield, Illinois, if one discounts the pre-Christmas rise in school absenteeism, one can appreciate the simultaneous rise in school absenteeism and emergency room visits followed 2 weeks later by an increase in industrial absenteeism. The initial trend was signaled 2 weeks prior to the peak by a 10% increase in emergency room visits (Figure 9). This pattern suggests that emergency room visits were a sensitive indicator of influenza activity in the area.

Fig. 8 EMERGENCY ROOM VISITS AND INDUSTRIAL ABSENTEEISM, COLUMBUS, OHIO, OCTOBER 28, 1972 – MARCH 3, 1973

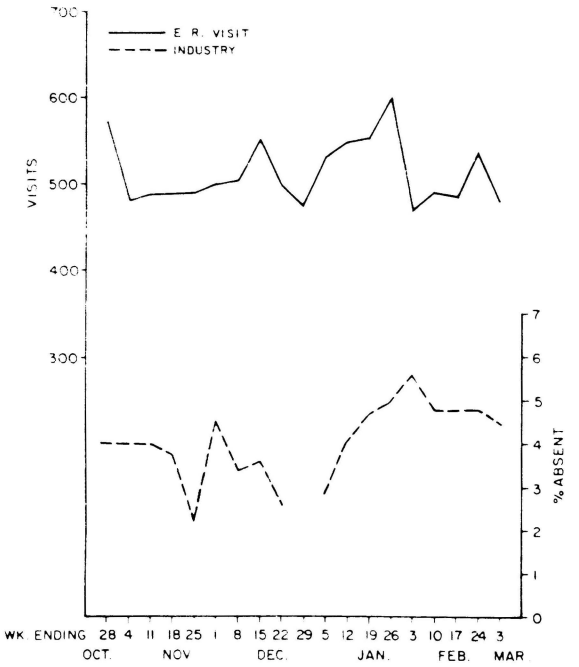
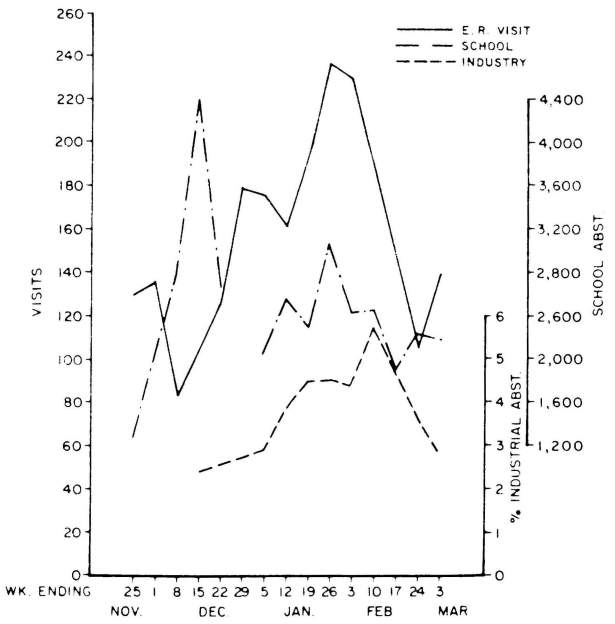
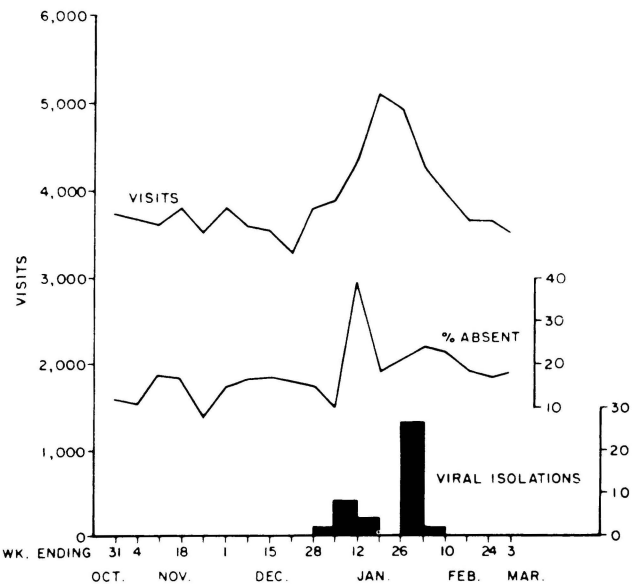


Fig. 9 EMERGENCY ROOM VISITS AND SCHOOL AND INDUSTRIAL ABSENTEEISM, ILLINOIS, NOV. 25, 1972 – MAR. 3, 1973



Georgia: In Atlanta, emergency room visits at Grady Memorial Hospital were monitored in conjunction with a program of obtaining specimens for viral isolation. In addition, industrial absenteeism was monitored in a large firm in Atlanta. Figure 10 shows the results of this surveillance system. It is clear that there was temporal relation between viral isolations and visits at the Grady emergency room indicating that the latter was a good index of influenza activity. It should be noted that during the week ending January 12, there was a severe ice storm in Atlanta which was probably responsible for increased industrial absenteeism.

Fig. 10 EMERGENCY ROOM VISITS, INDUSTRIAL ABSENTEEISM, AND VIRAL ISOLATIONS, GEORGIA, OCTOBER 31, 1972 – MARCH 3, 1973



Tennessee: A comprehensive surveillance system was undertaken in Shelby and Davidson Counties. In addition to the hospital and emergency room visits and school and industrial absenteeism, reported cases from private physicians in both counties were determined. Private physician reporting showed the best correlation with pneumonia-influenza data in the state (Figures 11 and 12).

Fig. 11 INFLUENZA SURVEILLANCE, SHELBY COUNTY, MEMPHIS, TENN., 1972-1973

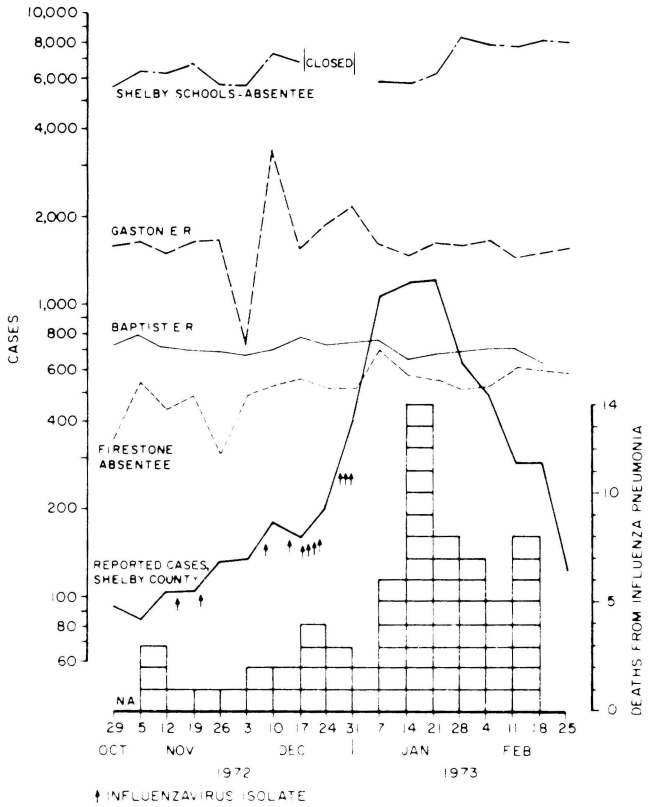
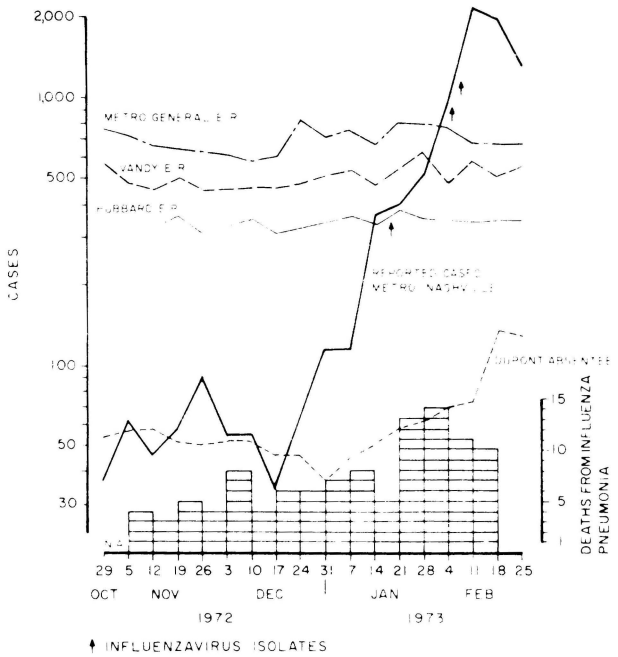


Fig. 12 INFLUENZA SURVEILLANCE, NASHVILLE AND METRO DAVIDSON COUNTY, TENN., 1972-1973

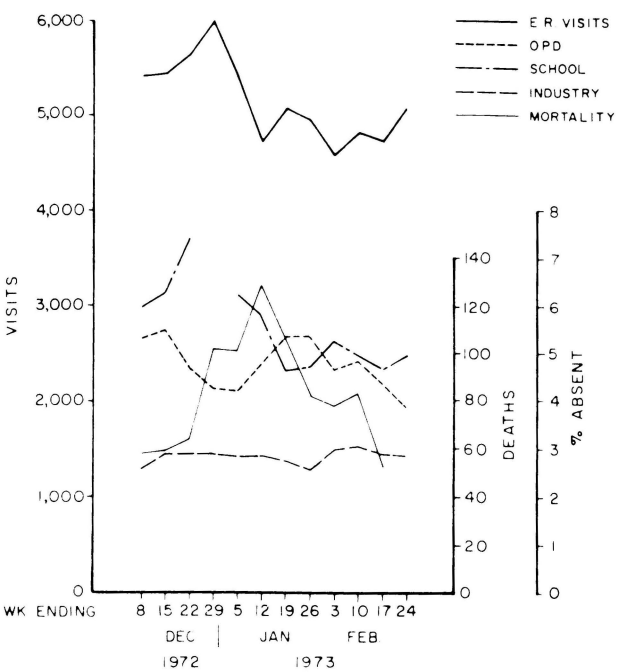


New York: Figure 13 illustrates that emergency room surveillance was a valuable marker of influenza activity in New York City. Industrial absenteeism did not vary significantly throughout the entire influenza season. However, emergency room visits peaked approximately 2 weeks prior to the peak incidence of mortality from pneumonia-influenza. The peak in school absenteeism for the 3rd week in December is difficult to interpret because of the Christmas vacation.

New Hampshire, Rhode Island, and Connecticut: School absenteeism remained relatively constant at about 6.5%. Emergency room and industrial absenteeism also remained stable. This corresponds with the appearance of only isolated outbreaks of influenza in these 3 states during 1972-1973.

In a number of states including Montana, North Dakota, South Dakota, Utah, Wyoming, New Mexico, and Oklahoma, no clear relationship between influenza activity and hospital emergency room

Fig. 13 EMERGENCY ROOM AND OUTPATIENT VISITS, SCHOOL AND INDUSTRIAL ABSENTEEISM, AND MORTALITY FROM PNEUMONIA-INFLUENZA, NEW YORK CITY, DECEMBER 8, 1972 - FEBRUARY 24, 1973



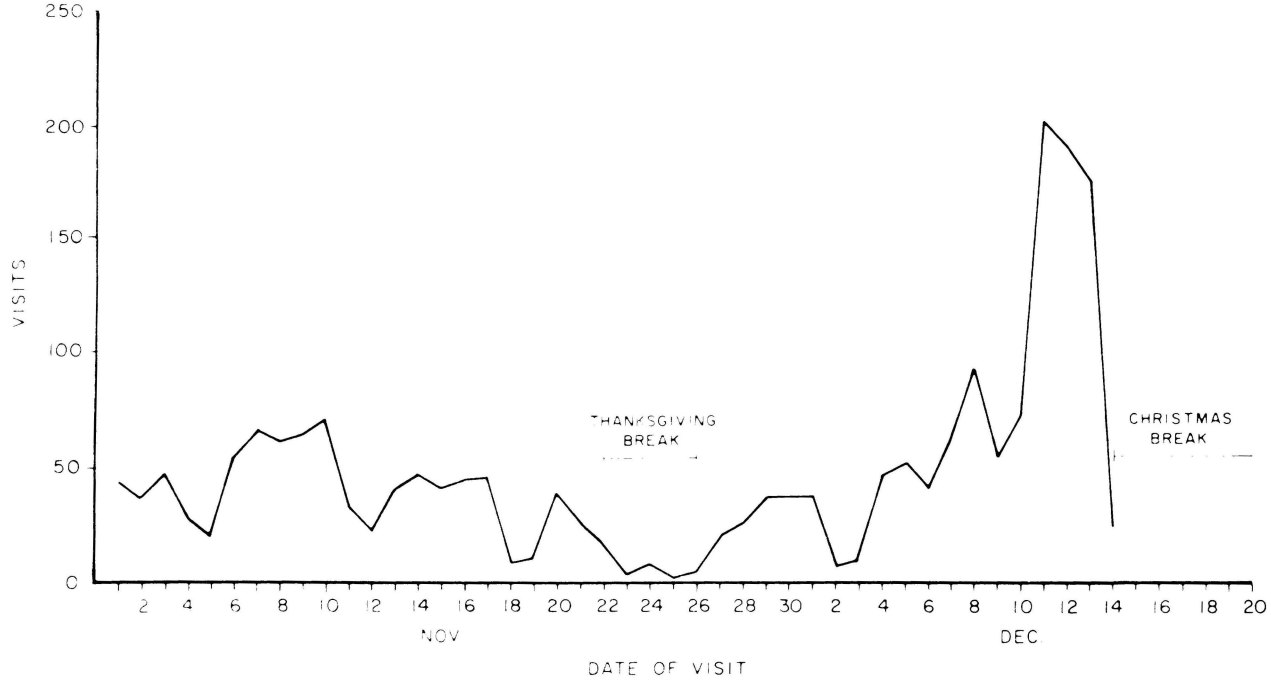
visits could be established. This is perhaps due in part to the fact that community hospitals in these areas do not serve as primary outpatient care facilities and consequently persons suffering from symptoms of influenza consult their family physician rather than seeking medical attention at a hospital emergency room. The addition of private physician reporting and viral isolation procedures to the standard parameters of school and industrial absenteeism would tend more accurately to reflect influenza-like activity in these areas.

Comment: It appears that on the state, county, and national level a variety of parameters must be monitored in order to characterize influenza-like disease during the influenza season. In urban areas where community emergency rooms are often used as entry points for primary medical care, monitoring of emergency room visits seems to represent a sensitive indicator of influenza-like disease within a community. In areas where this is not necessarily the case, private physician reporting, viral isolations, and industrial and school absenteeism often appear to be more sensitive indicators of disease activity. On either a state or regional level, combinations of these parameters must be used in order to reflect effectively the time, location, and severity of influenza activity within these areas.

IV. EPIDEMIC INVESTIGATIONS OF INFLUENZA OUTBREAKS

Pennsylvania: During the first 2 weeks of December 1972, a large outbreak of influenza occurred at the Keystone Job Corps Center for Women in Drums, Pennsylvania. Between 50-80% of the 542 girls living at the Center became ill during this time. By December 8, a 4-fold increase in infirmary visits associated with cough, fever, and sore throat was noted. On December 12, M. E. Matsko, M.D., Director of Health Services, notified the Pennsylvania Department of Health. Interviewing of girls and screening of infirmary records revealed that 288 girls were known to have been sick with upper respiratory infection between December 4 and December 13, 1972 (Figure 14).

Fig. 14 TOTAL INFIRMARY VISITS FOR UPPER RESPIRATORY INFECTION, KEYSTONE JOB TRAINING CENTER, DRUMS, PENNSYLVANIA, 1972



At least 38 girls from 7 different dormitories had onset of illness on or before December 4; histories obtained from these girls indicated that 10 had onset of illness while visiting away from the Center in 7 different cities:

Baltimore, Maryland	3
New York, New York	2
Buffalo, New York	1
Newark, New Jersey	1
Philadelphia, Pennsylvania	1
West Chester, Pennsylvania	1
Charlotte, North Carolina	1

It is interesting to note that at the time of the investigation 3 of these cities (Baltimore, New York, and Philadelphia) had already reported isolates of influenza A in 1972.

Personnel absentee data at the Center showed a 2- 3-fold increase on December 11-13. Surveillance data from Hazelton and Wilkes-Barre did not show any increased absenteeism in schools or industry at the time of the investigation.

Subsequent viral cultures revealed influenzavirus similar to A/England/42/72.

(Reported by M. E. Matsko, M.D., Director of Health Services, Keystone Job Corps Center for Women, Drums, Pennsylvania; W. D. Schrack, Jr., M.D., Director, Division of Communicable Diseases, Pennsylvania Department of Health; and David Rimland, M.D., EIS Medical Epidemiologist.)

Utah: A/England/42/72 influenzavirus was prominent in Utah in 1973. Activity in the state increased markedly after the Christmas holidays. Epidemics occurred at Utah State University, Brigham Young University, and in many small communities throughout the state. Surveillance data in Salt Lake County suggest that there was moderate influenza activity in late January and early February.

The following epidemic coincided with a statewide rise in influenza activity:

Between January 7 and 22, 1973, 26 cases of influenza due to A/England/42/72 occurred in a 42-bed chronic disease ward of a Utah hospital. The symptoms in the 22 patients and the 4 ward nurses included cough, fever, sore throat, and muscle aches. Diarrhea was present in 41% of the cases. The clinical attack rate was 67% (26/39). Six deaths occurred during the epidemic.

Influenza A virus was isolated from embryonated eggs in 1 patient's throat washing. No virus was isolated from stool cultures of 6 patients with diarrhea. Serologic evidence for A/England/72 infection was demonstrable in 62% (21/34) of paired acute and convalescent bloods. Five of the 6 patients who died did not have a convalescent blood specimen taken but are considered cases.

All patients were males. The median age of the ill population was 78, compared with the total population median age of 77. None of the patients had received an influenza immunization or booster that winter. Four patients demonstrated greater than 4-fold titer rises but did not have clinical evidence of influenza.

It is most likely that a visitor introduced the virus into the ward. The index case occurred in an 84-year-old man in a 24-bed dormitory room in 1 wing of the ward. Subsequent cases occurred in patients in both dormitory and semi-private rooms; influenza attack rates by sleeping room were similar. A dining room and a sun room, common to all patients, facilitated person-to-person transmission. Four members of the ward nursing staff also became ill with influenza-like symptoms during the epidemic. They had worked closely with all of the patients and probably served as transmitters of the virus.

(Reported by James R. Everett, M.D., EIS Officer, Sego Matsymiya, Nurse Epidemiologist V.A. Hospital, Salt Lake City; Taira Fukushima, M.D., Director, Bureau of Disease Prevention; and Lyman J. Olsen, M.D., M.P.H., Director of Health, Utah State Division of Health.)

North Dakota: During the last 3 weeks of January 1973, large numbers of students at the University of North Dakota in Grand Forks presented with upper respiratory

complaints consisting of headache, fever, chills, myalgia, pharyngitis, nonproductive cough, and oral temperature of 100-103°F. Approximately 30 students required hospitalization for dyspnea, hemoptysis, or severe prostration. Death occurred in 1 of the cases -- a 23-year old student with muscular dystrophy; progressive pneumonitis was unresponsive to broad spectrum antibiotics.

Subsequently, throat swabs and acute and convalescent sera confirmed the presence of A/England/42/72 (H3N2). The incidence of influenza-like illness in the remainder of Grand Forks during the University epidemic remained normal for that time of year. School absenteeism, hospital admissions, and private physician reporting revealed no increase in disease activity.

(Reported by J.A. Swenson, M.D., Director, University of North Dakota Student Health Service; A. A. Gustafson, M.P.H., Director, Grand Forks Public Health Laboratory; Kenneth Mosser, State Epidemiologist, J. R. Amos, M.D., State Health Officer, and Paul Larsen, M.D., EIS Officer, North Dakota State Department of Health.)

Georgia: An outbreak of influenza affecting more than 20% of the students at North Georgia College in Dahlonega occurred in early January 1973. An influenza-like illness was noted January 3 among students returning to campus from the Christmas holidays.

Influenza vaccine had been offered to students in October 1972, but many students failed to receive vaccination in either 1971 or 1972. Questionnaires answered by 75.1% of the male students and 36.5% of the female students, revealed that attack rates between previously immunized and unimmunized groups were not significantly different. Vaccination appeared to have little effect in reducing the severity of illness. Illness lasting more than 7 days occurred in 32.5% of those who did not receive influenza vaccine in 1972 and in 45.5% of those who received immunizations. A/England/42/72 was subsequently isolated from 4 of 6 throat swabs.

(Reported by John McCroan, Ph.D., Supervisor, Epidemiology Section, Department of Human Resources, Thomas W. McKinley, Senior Epidemiologist, Epidemiology Section, John M. Smith, B.S., Assistant Epidemiologist, and Maurice Miot, M.D., Chief, Virology Laboratory, Georgia Department of Human Resources.)

Missouri: A total of 181 cases of influenza occurred among 367 girls at a training center in Clay County, Missouri, during the second week of January 1973. Attack rates by area of residence within the complex revealed a higher attack rate (64%) in the orientation dormitory, while the remainder of the trainees experienced an attack rate of 47%. This difference was felt to reflect the greater personal contact in the orientation group.

Twenty girls with onset of symptoms within 48 hours of examination were selected at random and interviewed. Serum and nasopharyngeal swabs were obtained. Smears of nasal secretions stained with fluorescein-labeled anti-influenza A antibody provided rapid confirmation of the diagnosis of influenza.* In each of the 20 cases, virus isolation and/or serum antibody rise confirmed the diagnosis. Influenza A virus was isolated from 15 of the 20 nasopharyngeal cultures obtained. One of the isolates was further characterized as A/England/42/72.

Acute and convalescent sera were available on 14 of the 20 cases (Table 2). While 8 of the 14 had low initial hemagglutination inhibition (HI) antibody titers ($\leq$:10) against A/Hong Kong/8/68 (A/HK/68) antigen, all 14 had initial low titers to the A/England/42/72 (A/Eng/72) antigen. Pre-existing antibody titers of $\geq$1:40 to A/HK/68 did not prevent a typical illness in 4 cases, however, recovery of the virus was more frequent when pre-existing antibody was absent. Thirteen of the 14 patients had 4-fold or greater antibody rises to A/Eng/72, while only 11 of 14 responded with 4-fold or greater rises against A/HK/68. One case (case 11) had no antibody rise to either A/Eng/72 or A/HK/68, although she had a typical clinical illness and influenzavirus was recovered from the nasopharynx.

*Performed in the laboratory of Chien Liu, M.D., Kansas University Medical Center, Kansas City, Kansas

Table 2

Reciprocal HI Antibody Titers to A/Hong Kong/68 and A/England/72 Antigens in 14 Cases of Influenza Clay County, Missouri

Case	A/Hong Kong/68		A/England/72		Influenza A virus isolated
	S_1	S_2	S_1	S_2	
1	<10	20	<10	40	+
2	<10	40	<10	40	+
3	<10	80	<10	160	+
4	<10	40	<10	80	+
5	<10	20	<10	40	-
6	<10	20	<10	40	+
7	10	80	<10	40	-
8	10	160	<10	80	+
9	20	160	10	160	+
10	20	80	<10	40	+
11	40	40	10	20	+
12	40	80	10	40	-
13	80	320	10	160	-
14	160	160	<10	80	-

S_1 = Acute serum
S_2 = Convalescent serum
+ = Virus isolated
- = Virus not isolated

(Reported by Robert S. Schultz, M.D., Director, Clay County Health Department; Robert C. Kane, M.D., Medical Epidemiologist; Wyatt E. Rousseau, M.D., EIS Officer, Ecological Investigations Program; Gary R. Noble, M.D., Chief, Virus Disease Section, Ecological Investigations Program, Kansas City, Kansas; Herbert R. Domke, M.D., Director of Health, and H. Denny Donnell, Jr., M.D., Director, Bureau of Communicable Diseases, Missouri Department of Public Health and Welfare.)

V. WORLDWIDE INFLUENZA SURVEILLANCE

A. Influenza A

Australia: Australia reported outbreaks in both metropolitan areas and in military recruit camps from August to mid-September 1972. Disease was mild in severity with few fatalities. Attack rates varied from approximately 20% in the Sidney police force to only sporadic cases in Queensland. Strains of both A and B were noted. Influenza A isolates were similar to A/England/42/72.

Hong Kong: Local outbreaks of influenza A in the general population were noted from mid-August to September.

Thailand and Viet Nam: Both Thailand and Viet Nam reported outbreaks of influenza A. In Saigon, school absenteeism during the influenza season from September to October reached approximately 35%. Strains of virus A antigenically related to A/England/42/72 were isolated.

USSR: Influenza was widespread throughout the USSR. Scattered foci of influenza activity first appeared in October and November 1972. By January 1973, a widespread epidemic involving the general population occurred in Moscow and Leningrad where 70,000 and 30,000 cases, respectively, were reported daily. The estimated attack rate in Moscow in January 1973 was 18%.

Eastern Europe: More than 170,000 cases of influenza-like disease were reported in the Sofia area of Bulgaria in an epidemic which peaked November 27, 1972. Yugoslavia, Romania, Hungary, and Poland also reported widespread outbreaks of influenza with strains antigenically related to A/England/42/72.

Western Europe: France, the Federal Republic of Germany, and the Netherlands reported outbreaks of influenza A. Serologic sampling in France revealed attack rates which varied from greater than 25% in the Paris area to less than 10% in the Lyon area. Switzerland, Spain, and Italy also reported widespread outbreaks in late January and February 1973, with viruses similar to the A/England/42/72.

United Kingdom: Widespread outbreaks of influenza were reported in the United Kingdom. Approximately 71 strains of type A virus all similar to the A/England/42/72 variant were isolated. Total deaths and bed claims exceeded 1971-1972 levels.

B. Influenza B

The isolation of influenza B viruses from sporadic cases occurring in Hong Kong in December 1972 and January 1973 were reported in the WHO Weekly Epidemiological Record, Week 13, 1973. These strains were found to contain hemagglutinins showing marked antigenic changes from the viruses isolated previously. Influenza B viruses similar to those isolated in Hong Kong were also recovered from sporadic cases in Australia. During the same period influenza strains antigenically intermediate between the B/Hong Kong/5/72 and the previously prevalent strains were isolated in Germany and from a severe epidemic in a boarding school in England. In late March additional intermediate strains were isolated from local outbreaks in Germany.

Japan: From April to July 1973, extensive outbreaks occurred in nearly all regions of Japan with both "older," (i.e., similar to B/Massachusetts/1/71 or B/Vict/98926/70) and intermediate strains of influenza B being noted. In most areas, virus antigenically close to strains intermediate between the new variant B/HK/5/72 and previous B strains were noted. In Tokyo, Kanagawa, and Tochigi prefectures, B viruses similar to B/Vict/98926/70 were also isolated. A serologic survey done before the epidemic among children in Ibaiagi prefecture indicated that most did not possess antibodies against the B virus which caused the epidemic, whereas antibodies to previous B viruses were found in the same population.

United Kingdom: In the United Kingdom, as the winter outbreak of influenza A died down in January 1973, influenza B infection began to appear. A small but widespread epidemic of influenza B was identified mainly in institutionalized children in Southwest, Middle, and Northern England. Three different antigenic variants of virus were detected. Early in the year, isolates of influenza B strains similar to previous strains were isolated. In mid-March an outbreak in a boys' boarding school revealed antigenically "intermediate" strains of influenza B. In late May strains indistinguishable from B/HK/5/72 were isolated. In June, the "intermediate" and "old" strains began to disappear and only strains similar to B/HK/5/72 were isolated.

Antisera positive to previously prevalent strains failed to react significantly with the strains resembling B/Hong Kong/5/72, although such antisera reacted to some degree with the intermediate strains from England and Germany. B/Hong Kong/5/72 antisera also inhibited the European strains, but not the "older" strains. Preliminary studies indicate that the antigenic variation involves only the hemagglutinin and not the neuraminidase. Serologic surveys of young adults in England and in the United States showed that fewer than 20% in England and none in the United States had HI antibody titer of $\geq$1:40 to the B/Hong Kong/5/72 strain. Such findings suggest that these populations have had little experience with influenza B viruses resembling B/Hong Kong/5/72. The recovery of B/Hong Kong/5/72-like strains from Hong Kong, Australia, Japan, and England indicate that these strains are becoming prevalent.

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VI. LABORATORY REPORT

Influenza Laboratory Surveillance in the United States

In November 1972, virologists in the WHO collaborating laboratories in the United States were asked to report on a weekly basis the results of their influenza diagnostic tests. The number of specimens tested and the number of influenza cases confirmed by either serologic testing or virus isolation were reported for a 14-week period beginning November 17, 1972, and ending February 17, 1973. Approximately 40 laboratories reported recovery of 1,029 influenzaviruses, and 835 diagnostic, i.e. $\geq$4-fold, rises in hemagglutination-inhibition (HI) or complement fixation (CF) antibody titers occurred (Table 3).

Table 3

Influenza Laboratory Surveillance for the United States
November 1972 - February 1973

Week Ending	Number of Laboratories Participating	Viral Isolation			Paired Sera		
		Number Tested	Number Isolates A*	Number Isolates B**	Number Tested	Number Positive A*	Number Positive B**
11-17-72	16	92	2	1	157	0	0
11-24-72	26	122	2	0	225	6	0
12-01-72	39	279	1	3	343	1	0
12-08-72	32	235	14	8	261	0	0
12-15-72	42	392	6	2	178	4	0
12-22-72	39	349	29	0	171	8	2
12-29-72	37	288	12	5	114	5	0
1-06-73	42	488	66	3	306	12	0
1-13-73	45	670	103	11	427	51	0
1-20-73	49	678	135	0	417	60	0
1-27-73	46	891	201	8	361	101	1
2-03-73	45	537	147	2	535	147	0
2-10-73	43	808	155	1	764	255	0
2-17-73	38	460	112	0	594	182	0
TOTAL		6,289	985	44	4,853	832	3

*Influenza type A

**Influenza type B. Influenza B activity was documented in California, Colorado, Hawaii, Oregon, Washington, and Wisconsin during the period of surveillance.

During the 1972-1973 influenza season, diagnostic serology at CDC was routinely performed by HI tests using A/HongKong/8/68 (H3N2), A/England/42/73 (H3N2), and B/Victoria/98926/70 as antigens, and by CF tests using type A and type B ribonucleoprotein antigens. A total of 132 diagnostic (≥4-fold) rises to influenza A were obtained by HI and/or CF tests (Table 4). The Hong Kong and England antigens were almost equally effective in detecting HI antibody rises in paired sera. Of the total rises demonstrated, 92% were detected with Hong Kong antigen and 89% with England antigen. The CF test was less efficient and detected only 72% of the total rises. The Hong Kong HI test detected 14 diagnostic rises that were missed by the other 2 tests, and the England HI test detected 10 rises that were missed by the other tests. None of the CF rises were missed by both HI antigens.

Table 4

Results of Hemagglutination Inhibition (HI) and Complement Fixation (CF) Tests for the Serodiagnosis of Influenza A (1972-1973)

Serologic Test	Antigen(s)	Diagnostic Rises* No.	Percent
HI and/or CF (Totals)		132	100
HI	A/Hong Kong/8/68 (H3N2)	122	92
HI	A/England/42/72 (H3N2)	118	89
CF	Influenza A ribonucleoprotein	95	72
HI	A/Hong Kong/8/68 only	14	11
HI	A/England/42/72 only	10	8
CF	Influenza A ribonucleoprotein only	0	0

*≥4-fold rise in antibody titer

Geographical Distribution and Antigenic Analysis of Isolates

From July 1972 to July 1973, the International Influenza Center for the Americas examined 489 influenza A and 43 influenza B strains which were sent from 30 laboratories in the United States and 17 laboratories in other countries (Table 5).

Type A: The majority of A strains tested are antigenically close to A/England/42/72 (H3N2) (Table 3). A single strain from Alaska isolated in August 1972 is more closely related to the A/Hong Kong/5/72 variant. Strains recovered in August and September 1972 from Buenos Aires (5) and Chile (1), and in January 1973 from Brazil (7) resemble the previous A/Hong Kong/68 strains.

Hemagglutination inhibition reactions from representative 1972-1973 influenza A viruses are shown in Table 6. Except for A/Hong Kong/5/72 and A/Rio/1/73, the 1972-1973 strains form a homogeneous group of strains closely related to A/England/42/72. Most A strains are inhibited by A/Hong Kong/68 reference antisera with titers at least 4-fold less than the homologous titers. These strains generally show less than 4-fold of homologous titer in reactions with A/Hong Kong/5/72 antiserum.

Table 5

Influenza A Isolates[1] Examined July 1972 - June 1973

Geographic Origin	No. of Strains
North America	
AL	3
AK	24[2]
AZ	1
AR	12
CA	5
CO	18
CT	1
FL	4
GA	13
ID	10
IL	26
IN	2
IA	1
KS	9
KY	9
LA	7
MA	2
MD	9
MI	4
MN	7
MS	7
MO	2
NC	1
NH	7
NJ	3
NY	18
OH	8
OK	14
OR	9
PA	28
SC	3
TN	5
TX	8
UT	1
VA	17
WA	31
WI	10
WY	2
Nassau	5
TOTAL	346

Geographic Origin	No. of Strains
Caribbean	
Canal Zone	6
Jamaica	7
Trinidad	37
South America	
Argentina	12[3]
Brazil	19[4]
Chile	13[5]
Uruguay	3
TOTAL	97

Geographic Origin	No. of Strains
Western Pacific	
Australia	3
Fiji	2
Guam	10
Hawaii	13
Philippines	1
Taiwan	3
Thailand	2
Tokyo	3
Yap	9
TOTAL	46

[1]All strains are antigenically close to A/England/42/72 (H3N2) unless otherwise indicated.
[2]A/Alaska/16/72 (August 1972) resembles A/Hong Kong/5/72 (H3N2) antigenically.
[3]Five strains from Buenos Aires (August 1972) resemble A/Hong Kong/8/68.
[4]Seven strains from Rio de Janeiro (January 1973) resemble A/Hong Kong/8/68.
[5]A/Chile/239/72 (September 1972) resembles A/Hong Kong/8/68.

Table 6

Hemagglutination Inhibition Reactions* Among 1972-1973 Influenza A Viruses

Virus Strains	Antisera* A/Aichi/2/68(H3)**	A/HK/68(H3)-eq/56(Heq1)	A/Hong Kong/68	A/Eng/72(H3)-eq/56(Heq1)	A/England/72	A/HK/5/72	A/Victoria/4/72	A/Brooks/4/72	A/Georgia/3/73	A/Oregon/4/73
A/Hong Kong/8/68	7240	640	640	40	113	80	226	453	320	320
A/England/42/72	1810	160	160	226	320	80	320	905	1810	640
A/Hong Kong/5/72	453	80	80	57	80	453	80	320	160	226
A/Alaska/15/73	1810	160	160	160	453	160	640	905	1280	905
A/Ann Arbor/1/73	2560	113	113	160	320	80	226	905	905	905
A/Brooks AFB/4/72	2560	160	160	226	320	80	453	1280	905	1280
A/Canal Zone/5/72	1810	160	113	160	320	113	226	1280	640	905
A/Chile/304/72	2560	160	160	160	320	113	453	1280	905	1280
A/Colorado/2/73	3620	160	226	453	453	160	640	1280	1280	1280
A/Georgia/3/73	1810	113	113	160	160	80	453	640	640	640
A/Hawaii/13/72	1810	160	160	160	320	113	453	905	905	1280
A/Jamaica/2/73	2560	160	160	160	320	28	320	905	1280	640
A/Kansas/3/73	2560	113	160	160	320	113	320	905	1280	1280
A/Nassau/2/73	2560	160	160	226	226	28	320	1280	905	1280
A/NYC/1/72	1810	113	113	113	320	113	453	640	453	640
A/Oregon/4/73	2560	113	113	320	226	113	453	905	905	905
A/Pennsylvania/6/73	2560	113	80	160	160	28	160	640	640	905
A/Rio/1/73	3620	453	453	28	80	40	113	320	226	320
A/SUNY/1/73	1810	80	113	160	320	113	453	1280	640	905
A/Tokyo/1/73	2560	160	160	320	320	320	113	453	1280	1280
A/Trinidad/15/73	2560	160	160	160	320	57	453	1280	905	905
A/Washington/19/73	2560	160	160	226	453	226	453	1280	905	1280
A/Wisconsin/3/73	3620	160	113	160	320	160	453	1280	905	1280
A/Victoria/4/72	2560	160	226	320	320	160	453	1810	1280	1280

*Geometric mean titers of duplicate HI tests.
*Chicken antisera treated with 100 units RDE.
**WHO reference antiserum prepared against electrophoretically isolated H3 hemagglutinins of the recombinant virus A/Aichi/2/68(H3)-Bel/42(N1).
Shading indicates 4-fold or greater differences from homologous antiserum titer.

Twelve influenza A strains were tested by neuraminidase inhibition (NI) with antiserum against recombinant influenzavirus A/equine/Prague/1/56 (Heq1)-England/42/72(N2) (Table 7). (Since there is no recognized antigenic relationship between equine/Prague hemagglutinin and known human influenza strains, the presence of Heq1 antibodies in the reference sera should not interfere in the NI reaction.) Five strains show less than a half-log difference from the NI titer of A/England/42/72 and only 1 strain has more than 1 log difference.

Table 7

Differences Between Neuraminidase Inhibition (NI) Titers of 1972-1973 Influenza A viruses and A/England/42/72 (H3N2) with Anti-N2 Chicken Serum*

Negative Log_{10} Difference ≤0.5	0.6 - 1.0	1.1 - 1.5
A/Nassau/2/73	A/New York City/1/72	A/SUNY/1/73
A/Pennsylvania/8/73	A/Kansas/3/73	
A/Chile/304/72	A/Tokyo/1/73	
A/Victoria/4/72	A/Wisconsin/3/73	
A/Jamaica/2/73	A/Brooks/4/72	
	A/Rio de Janeiro/1/73	

*Chicken antiserum prepared against recombinant influenzavirus A/equine/Pr/1/56(Heq1)-England/42/72(N2).

Type B: Antigenic analyses of the 43 influenza B isolates received in 1972-1973 indicate that some strains have undergone antigenic variation involving the hemagglutin but not the neuraminidase antigen. The hemagglutination inhibition reactions of 11 representative B viruses and the WHO reference 1970-1971 strains are shown in Table 8. The hemagglutinin antigens of the B/Hong Kong strains have undergone a major shift from those of previous strains. B/Hong Kong and the 1970-1971 B strains do not cross-react significantly. The intermediate strains from Buenos Aires, Hannover, Hawaii, Minnesota, and Washington cross-react with the 1970-1971 strains and to some extent with the Hong Kong strains.

Table 8

Hemagglutination Inhibition Reactions of 1972-1973 Influenza B Viruses with Reference Chicken Antisera

	Antisera						
Influenza B Strains	B/Vic	B/Mass	B/HK/72	Anti H*	B/HK/73	B/Haw	B/Hann
B/Arizona/1/73	320	320	<	<	<	80	80
B/Berkeley/1/73	640	160	<	<	<	80	80
B/Buenos Aires/3/73	320	640	10	ND	20	320	320
B/U. Chicago/1/73	80	160	<	<	<	80	40
B/Hannover/3/73	160	160	40	20	20	640	320
B/Hawaii/3/73	320	160	80	20	20	320	160
B/Hong Kong/5/72	<	<	320	160	80	20	10
B/Hong Kong/8/73	<	<	160	320	320	40	20
B/Massachusetts/1/71	320	640	10	10	<	320	320
B/Mayo Clinic/8/73	160	160	20	10	20	40	40
B/Oregon/2/73	160	160	10	<	<	ND	320
B/Washington/1/73	320	160	80	20	10	320	160
B/Victoria/98926/70	640	80	<	<	<	160	80

*Prepared against B/Hong Kong/5/72 virus heated for 2 hours at 56° to destroy the neuraminidase.
<means less than 1:10. ND means not done.

Hemagglutination inhibition tests indicate that the influenza B strains comprise 3 antigenic groups (Table 9):

1. Twenty-four strains show only minor variation in hemagglutinin from that of the B viruses prevalent in 1970-1971.

2. B/Hong Kong/5/72 and B/Hong Kong/8/73 show marked changes in hemagglutinin antigens from the previous prevalent strains.

3. Seventeen influenza B isolates which are antigenically intermediate between the Hong Kong strains and the previous prevalent strains were received from Germany (1), Hawaii (10), and Argentina (3) and, in the continental United States, from Minnesota (1) and Washington (2).

Table 9

Antigenic Grouping of Influenza B Isolates From July 1972 - June 1973 Based on HI Reactions With Strain Specific Antisera

Geographic Origin	No. of Isolates	70-71[1]	HK/72[2]	Intermediate[3]
Argentina	3			3
Arizona	11	11		
Australia	1	1		
California	1	1		
Germany	1			1
Hawaii	11	1		10
Hong Kong	2		2	
Illinois	1	1		
Minnesota	8	7		1
Oregon	2	2		
Washington	2			2
TOTAL	43	24	2	17

[1]Hemagglutinin antigen similar to B/Victoria/98926/70 and B/Massachusetts/1/71

[2]Hemagglutinin antigens of B/Hong Kong/5/72 and B/Hong Kong /8/73 show marked changes from the previous prevalent strains.

[3]Intermediate strains show antigenic crossing with 1970-1971 strains and some degree of inhibition by antisera to B/Hong Kong/5/72 and B/Hong Kong/8/73.

The neuraminidase antigens of the influenza B viruses have not changed. Table 10 shows neuraminidase inhibition reactions of B strains isolated in 1966, 1971, and 1972. Each antiserum inhibits all of the strains and heterologous titers are less than 2-fold different from homologous titers.

Table 10

Reciprocal Neuraminidase Inhibition Reactions of Influenza B Viruses

Virus Strain	Antisera B/Mass/66(N)*	B/Mass/71**	B/HK/72**
B/Massachusetts/3/66	355	320	178
B/Massachusetts/1/71	320	355	200
B/Hong Kong/5/72	200	563	224

*Monospecific rabbit antiserum prepared against purified neuraminidase isolated from B/Massachusetts/3/66 virus.
**Strain specific chicken antisera.

VII. METHOD FOR DIAGNOSIS OF INFLUENZA OUTBREAKS

Two principal procedures are available to establish the occurrence of influenza: 1) isolation of the virus and 2) a rise in titer of influenza antibody between acute and convalescent serum specimens.

As the public generally believes all febrile upper respiratory disease is the "flu," the isolation of the influenzavirus is important. The diagnosis of influenza must initially be made either serologically or by virus isolation. Facilities for such diagnosis are available in almost every state and large city, and private practitioners are encouraged to use these facilities if they suspect an outbreak of influenza. Only when a virus has been isolated during an outbreak can the type of influenzavirus causing the outbreak and its relationship to previous types be established with certainty. Even though multiple virus isolates obtained from the same epidemic will undoubtedly confirm that the epidemic is caused by a specific influenzavirus, virus isolation is neither a convenient nor practical means of laboratory documentation of epidemics. Theoretically, it should be possible to isolate and identify an influenzavirus in as little as 48 hours; but, in practice, it may take a week or more before an isolate is identified. Multiple blind passages of virus may be required before an isolation is made. Finally, it is much easier to demonstrate a diagnostic rise in antibody than to isolate a virus from a single infected person.

Serologic diagnosis of influenza infection is most readily made by the hemagglutination-inhibition (HI) or by the complement fixation (CF) tests. CF or HI tests can be run within a 24-hour period; however, there is a considerable time lag in making a serologic diagnosis since collection of acute and convalescent sera from the same individual takes 2 to 3 weeks. To minimize this time lag, a number of investigators (1, 2, 3) have compared groups of acute and convalescent sera taken from 1 epidemic, but from different persons.

By the time the presence of an epidemic has been established, there are usually a number of individuals in the community who are already convalescent from the illness, while a number of other persons are in the early acute stages. At one point in time, 10 or more acute specimens and 10 or more convalescent specimens can easily be collected. Since influenza antibody levels vary by age and by influenza vaccination status, the acute and convalescent groups should be equivalent with respect to age and preferably consist of unvaccinated individuals.

The same serologic test (CF or HI) is performed in a single run on each of the sera in each group. Geometric mean titers are then calculated for the acute and the convalescent groups. Although for any single individual, a 4-fold rise in titer constitutes a diagnostic rise, a 4-fold rise in geometric mean titer is clearly too stringent a criterion for documentation of an epidemic: for example, if 6 to 10 persons involved in the same outbreak had exactly a 4-fold rise in influenza antibody and the remaining 4 had no rise, one would not hesitate to make the diagnosis of an influenza outbreak even though the geometric mean titer rise for the group of 10 was less than 4-fold.

The statistical significance of a comparison between acute geometric mean titers (GMT) and convalescent GMT must be made by using log titers because of the marked non-normality of titer data. A conventional student's t test is then performed on the log titers.

The comparison of acute and convalescent sera can apply to most epidemic illnesses for which a diagnosis can be made serologically. In instances where acute specimens are not available, one may be tempted to compare persons who did not become ill with persons who are convalescent. It may be possible, however, that persons who did not become ill may have had pre-existing high titers and not have become ill because they were already immune to the agent. In this event, the "not ill" group will have a high geometric mean titer and will not differ significantly from the convalescents.

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VIII. RECOMMENDATION OF THE PUBLIC HEALTH SERVICE ADVISORY COMMITTEE ON IMMUNIZATION PRACTICES

INFLUENZA VACCINE

INTRODUCTION

Influenza occurs to some extent in the United States every year, but its incidence and the areas affected are quite variable. Periodically, influenza appears in epidemic form. This seems to occur when the antigens of prevalent influenzaviruses change sufficiently to render the population susceptible. Type A and type B influenzaviruses both undergo changes in their antigens. Such changes usually occur gradually, but they can be rapid and abrupt. Epidemics caused by type A influenzaviruses are more frequent and are generally more severe than those caused by type B.

Inactivated influenza vaccines have not been uniformly effective in the past, and whatever protection they afforded was relatively shortlived. Current vaccines contain more antigen than products available before 1972 and should provide good protection against influenza when the prevalent viruses are identical or similar to those in the vaccine.

Influenza vaccine should be given to chronically ill patients and to older persons in general. These 2 groups appear to be at greatest risk of becoming severely ill with influenza. Because some influenza occurs every year, annual vaccination of "high-risk" patients is indicated as a routine procedure regardless of the amount of influenza expected in any specific geographic area.

INFLUENZAVIRUS VACCINES

Bivalent Vaccine*

The Bureau of Biologics, Food and Drug Administration, reviews influenza vaccine formulation regularly and recommends reformulation with contemporary antigens when indicated. Bivalent influenza vaccine this year will contain a new type A influenzavirus representative of currently prevalent "England" strains. Each adult dose of the 1973-74 vaccine will contain not less than 1000 chick cell agglutinating (CCA) units of antigen in the following proportion: 700 CCA units of a type A strain comparable to the prototype, A England/42/72(H3N2),** and 300 CCA units of a type B strain, B Massachusetts/1/71. Vaccines from all producers are highly purified and should be relatively free from adverse reactions.

Monovalent Type B Vaccine

Since late 1972, new strains of type B influenzavirus have been identified as the cause of characteristic influenza illness. They appeared first in Hong Kong in December 1972 and have since been recovered from influenza cases in Australia and England. It is too early to judge whether these strains will generally supplant currently prevalent type B viruses in the United States in the 1973-74 influenza season. However, it is reasonable to expect that they may become widely disseminated.

Since these type B antigens differ considerably from prior strains, little natural immunity to them can be expected to exist in the general population. Likewise, the available bivalent influenza vaccine cannot be expected to give optimal protection against them.

Anticipating the possibility that these type B influenzaviruses will become widely prevalent in the United States, the Bureau of Biologics prepared guidelines for production of a monovalent type B influenza vaccine containing an antigen representative of the new strains. This monovalent vaccine is expected to be commercially available prior to the 1973-74 influenza season. It should be used as a supplemental vaccine for optimal protection of persons at high-risk who are already recommended to receive bivalent vaccine.

VACCINE USAGE

General Recommendations

Annual vaccination is recommended for persons of all ages who have such chronic conditions as 1) heart disease of any etiology, particularly with mitral stenosis or cardiac insufficiency; 2) chronic bronchopulmonary diseases, such as asthma, chronic bronchitis, bronchiectasis, and emphysema; and 3) diabetes mellitus and other chronic metabolic disorders.

Annual vaccination is recommended for older persons because influenza outbreaks are commonly associated with excess mortality in older age groups.

Vaccinating persons who provide essential community services may also be considered if local priorities justify. However, before undertaking such programs, those responsible should take into account a number of reasonable constraints: difficulties inherent in predicting influenza epidemics, variability in vaccine effectiveness, cost, availability of vaccine, and the chance that vaccine will be diverted from persons with chronic illnesses who are at particular risk.

Schedule

The primary series of bivalent influenza vaccine has traditionally been 2 doses. Preliminary data indicate that with the more potent influenza vaccines available in recent years, the second dose provides little additional benefit. It is therefore reasonable to give a single dose of vaccine for either primary or annual booster vaccination. (Dose volumes for adults and children and the recommended route of administration are specified in the manufacturers' package labeling.)

A single dose of the supplemental monovalent type B influenza vaccine should follow and not be given simultaneously with bivalent vaccine. This is because the additional amount of antigen in the monovalent product might increase the chance of adverse reaction. Furthermore, separating the vaccines by 2 weeks or more might enhance an overall type B antibody response.

Influenza vaccination should be scheduled for completion by mid-November.

Precautions

Influenza vaccines are prepared from viruses grown in embryonated eggs and ordinarily should not be administered to persons clearly hypersensitive to egg protein, ingested or injected.

*Official name: Influenza Virus Vaccine, Bivalent

**The World Health Organization has recommended a revised system of nomenclature for type A influenzaviruses which includes their strain designation and a description of the 2 surface antigens, hemagglutinin (H) and neuraminidase (N).

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STATE EPIDEMIOLOGISTS

Key to all disease surveillance activities are those in each State who serve the function as State epidemiologists. Responsible for the collection, interpretation and transmission of data and epidemiologic information from their individual States, the State epidemiologists perform a most vital role. Their major contributions to the evolution of this report are gratefully acknowledged.

Alabama . . . . . Frederick S. Wolf, M.D.
Alaska . . . . . Donald K. Freedman, M.D.
Arizona . . . . . Philip M. Hotchkiss, D.V.M.
Arkansas . . . . . Stephen K. Felts, M.D., Acting
California . . . . . James Chin, M.D.
Colorado . . . . . Thomas M. Vernon, Jr., M.D.
Connecticut . . . . . James C. Hart, M.D.
Delaware . . . . . Ernest S. Tierkel, V.M.D.
District of Columbia . . . . . Donald K. Wallace, M.D.
Florida . . . . . Chester L. Nayfield, M.D.
Georgia . . . . . John E. McCroan, Ph.D.
Hawaii . . . . . Ned Wiebenga, M.D.
Idaho . . . . . John A. Mather, M.D.
Illinois . . . . . Byron J. Francis, M.D.
Indiana . . . . . Charles L. Barrett, M.D.
Iowa . . . . . Charles A. Herron, M.D.
Kansas . . . . . Don E. Wilcox, M.D.
Kentucky . . . . . Calixto Hernandez, M.D.
Louisiana . . . . . Charles T. Caraway, D.V.M.
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Massachusetts . . . . . Nicholas J. Fiumara, M.D.
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Mississippi . . . . . Durward L. Blakey, M.D.
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Nebraska . . . . . Paul A. Stoesz, M.D.
Nevada . . . . . William M. Edwards, M.D.
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New York State . . . . . Alan R. Hinman, M.D.
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North Carolina . . . . . Martin P. Hines, D.V.M.
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Ohio . . . . . John H. Ackerman, M.D.
Oklahoma . . . . . Stanley Ferguson, Ph.D.
Oregon . . . . . John A. Googins, M.D.
Pennsylvania . . . . . W. D. Schrack, Jr., M.D.
Puerto Rico . . . . . Angel M. Ayala, M.D.
Rhode Island . . . . . James R. Allen, M.D. (Acting)
South Carolina . . . . . William B. Gamble, M.D.
South Dakota . . . . . Robert S. Westaby, M.D.
Tennessee . . . . . Robert H. Hutcheson, Jr., M.D.
Texas . . . . . M. S. Dickerson, M.D.
Utah . . . . . Taira Fukushima, M.D.
Vermont . . . . . Geoffrey Smith, M.D.
Virginia . . . . . Robert S. Jackson, M.D.
Washington . . . . . Thieu Nghiem, M.D.
West Virginia . . . . . N. H. Dyer, M.D.
Wisconsin . . . . . H. Grant Skinner, M.D.
Wyoming . . . . . Herman S. Parish, M.D.

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U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
PUBLIC HEALTH SERVICE
CENTER FOR DISEASE CONTROL
ATLANTA, GEORGIA 30333

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